Interest in and reactions to genetic risk information: the role of implicit theories and self-affirmation

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Interest in and Reactions to Genetic Risk Information:

The Role of Implicit Theories and Self-Affirmation

Running title: Implicit theories and Self-affirmation

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Abstract

Rationale: Implicit theories reflect core assumptions about whether human attributes are malleable or fixed: *incremental theorists* believe a characteristic is malleable whereas *entity theorists* believe it is fixed. People with entity theories about health may be less likely to engage in risk-mitigating behavior. Spontaneous self-affirmation (e.g., reflecting on one's values when threatened) may lessen defensiveness and unhealthy behaviors associated with fixed beliefs, and reduce the likelihood of responding to health risk information with fixed beliefs. Method: Across two studies conducted in the US from 2012-2015, we investigated how self-affirmation and implicit theories about health and body weight were linked to engagement with genetic risk information. In Study 1, participants in a genome sequencing trial (n=511) completed cross-sectional assessments of implicit theories, self-affirmation, and intentions to learn, share, and use genetic information. In Study 2, overweight women (n=197) were randomized to receive genetic or behavioral explanations for weight; participants completed surveys assessing implicit theories, self-affirmation, self-efficacy, motivation, and intentions. Results: Fixed beliefs about weight were infrequently endorsed across studies (10.8-15.2%). In Study 1, participants with stronger fixed theories were less interested in learning and using genetic risk information about medically actionable disease; these associations were weaker among participants higher in self-affirmation. In Study 2, among participants given behavioral explanations for weight, stronger fixed theories about weight were associated with lower motivation and intentions to eat a healthy diet. Among participants given genetic explanations, being higher in self-affirmation was associated with less fixed beliefs. Conclusion: Stronger health-related fixed theories may decrease the likelihood of benefiting from genetic information, but less so for people who self-affirm.

Introduction

Implicit theories

Implicit theories, also called mindsets or lay theories, reflect core assumptions about the malleability of traits and characteristics (Dweck, 2006; Dweck & Leggett, 1988; Molden & Dweck, 2006). People who hold *incremental* or *growth* theories believe characteristics can change; people who hold *entity* or *fixed* theories believe they cannot. A person can endorse growth theories in one domain (e.g., artistic ability) and fixed theories in another (e.g., intelligence; Dweck et al., 1995; Molden & Dweck, 2006). Implicit theories guide how people self-regulate and respond to challenges (Burnette, O'Boyle, VanEpps, Pollack, & Finkel, 2013): people with growth theories respond to setbacks less helplessly and exert more effort than those with fixed theories. For example, people with health-related growth mindsets report greater behavior change self-efficacy, greater intentions to diet, more physical activity (Arciszewski et al., 2012; Lyons et al., 2013), and less avoidant coping (Burnette, 2010). Implicit theories can be changed through interventions, thus improving outcomes such as academic grades (Paunesku et al., 2015) or weight control (Burnette & Finkel, 2012).

We explored whether implicit theories influence how people engage with genetic/genomic information about disease. Genetic risk information is increasingly available (Collins & Varmus, 2015), yet translation into population health benefits has lagged (Khoury et al., 2007). Research is needed to examine who is interested in genetic risk information and how people react to causal information about disease. To date, no research has explored whether implicit theories are associated with attitudes and intentions concerning genetic risk information.

Implicit theories may influence how people engage with genetic risk information. Genetic information allows people to proactively cope with potential health threats (Aspinwall et al.,

2015; Aspinwall, Taber, Kohlmann, & Leachman, 2013), and people often expect that genetic information will improve health (Biesecker et al., 2014). However, people with fixed theories who believe health cannot change may not perceive benefit in learning genetic risk or changing behavior in response. Conversely, people with growth theories try harder when faced with challenges (e.g., learning of disease risk) and thus may seek genetic risk information and engage in preventive behavior. These hypotheses are consistent with limited prior evidence that people who perceived greater control over preventing or managing disease reported greater intentions to learn genetic risk information (Sweeney, Ghane, Legg, Huynh, & Andrews, 2014).

People might endorse a fixed theory if they ascribe genetic rather than behavioral causal attributions to a particular characteristic. Perceived genetic or behavioral causal attributions, beliefs about gene by environment interactions (Condit & Shen, 2011), fatalism (Shen, Condit, & Wright, 2009), and genetic determinism (Parrott et al., 2004) could be conceptually related to implicit theories and are related to engagement with genetic testing. We expected causal attributions and implicit theories to be related in that people who attribute disease to genetic factors should be higher in entity beliefs—and people who endorse gene-by-environment interactions should be lower in entity beliefs—but that implicit theories and specific beliefs about genetics would not be redundant. A person could think that health cannot change for reasons other than that health is caused by genetics, and most people do not endorse purely genetic causes or behavioral causes of disease (Nguyen, Oh, Moser, & Patrick, 2014). We examined the association of implicit beliefs with causal attributions and beliefs about gene by environment interactions.

Implicit theories might also influence how people respond to learning that genetics or behavior influences disease risk. One might expect that people who learn about genetic causes

would have lower intentions to change lifestyle behaviors (Senior & Marteau, 2007) and/or to adopt fixed theories about weight. Although some studies have shown genetic information about weight to be demotivating (Dar-Nimrod, Cheung, Ruby, & Heine, 2014; Persky, Ferrer, & Klein, 2016), receiving information that genes contribute to obesity does not typically decrease self-efficacy, intentions, or health behaviors (Conradt et al., 2009; Meisel, Beeken, van Jaarsveld, & Wardle, 2015; Persky & Street, 2015; Rief et al., 2007; Sanderson, Persky, & Michie, 2010). These mixed results suggest the importance of examining moderators of responses to risk information, such as implicit theories about body weight.

Risk and threatening health information

Learning about one's genetic risk for disease or genetic/behavioral causes of a health problem could be threatening if it confers bad news (i.e., high risk) or implicates the self as responsible for poor health. People may respond defensively to protect their self-integrity (Steele, 1988) such as by avoiding information or doubting its accuracy. People could also respond defensively by adopting fixed theories, which may either obviate the need to change their behavior (e.g., why diet if weight cannot change?), or provide an explanation that does not implicate the self for failed behavior change attempts (e.g., dieting failed because weight cannot change).

Some evidence supports the idea that fixed theories may promote defensive (i.e., self-protective) attitudes when people feel threatened. For example, people with fixed theories often respond to setbacks and challenges defensively by engaging in strategies to protect the self (e.g., withdrawing effort) because they fear others will notice their weaknesses (Dweck & Leggett, 1988). When people feel threatened, inducing growth beliefs can improve outcomes (Molden & Dweck, 2006). Further, a meta-analysis showed that implicit theories have a greater influence on

some aspects of self-regulation when people feel threatened, such as by failure feedback or setbacks (Burnette et al., 2013). More specifically, under conditions of threat as opposed to no threat, growth theories were more strongly associated with lower likelihood of setting performance goals and higher likelihood of setting learning goals, as well as lower likelihood of using helpless strategies and higher likelihood of using mastery strategies.

Self-affirmation

If fixed theories are associated with and can facilitate defensiveness, then self-affirmation might reduce effects and endorsement of fixed theories. When people are given the opportunity to reflect on core strengths and values ("self-affirmation"), they respond less defensively to self-threats (Epton, Harris, Kane, van Koningsbruggen, & Sheeran, 2014). People differ in how much they naturally self-affirm when feeling threatened, termed spontaneous self-affirmation (SSA; Cornil & Chandon, 2013; Harris, Griffin, Napper, Schuez, Stride, & Bond, 2017). Individuals higher in SSA may be less defensive in health contexts (Taber et al., 2016). We expected that people with fixed theories would express lower intentions to obtain potentially threatening genetic risk information, but that SSA would attenuate these associations. We also predicted that people who self-affirm will be less likely to defensively *adopt* fixed beliefs as a defensive strategy upon learning about explanations for obesity.

Current Research and Hypotheses

Two studies examined the role of health-related implicit theories in multiple aspects of learning and responding to genetic risk information in two disparate studies and samples (Figure 1). Specifically, we examined how implicit theories are associated with intentions to learn genetic risk information (Study 1), how implicit theories influence responses to receiving information about risk factors for disease (Study 2), and how learning risk information influences

endorsement of implicit theories themselves (Study 2). We also examined SSA, heretofore unexplored with respect to implicit theories.

<INSERT FIGURE 1 ABOUT HERE>

Study 1

We examined implicit theories about health among people facing potential receipt of actual genetic test results. We hypothesized that greater fixed theories would be associated with less proactive and adaptive responses. Proactive coping involves identifying stressors in advance and acting to reduce their impact (Aspinwall & Taylor, 1997). We examined multiple constructs broadly indicative of proactive and adaptive coping, including anticipated negative affect concerning high risk; intentions to learn risk information for medically actionable disease, nonactionable disease, and carrier status; intentions to share this information with relatives; and intentions to use information about medically actionable disease to change behavior. There may be benefits of learning risk for disease that is not medically actionable, such as improved knowledge, life planning, and hope (Chao et al., 2008; Williams et al., 2010), and learning carrier status might benefit relatives. Sharing genetic information could be proactive, as it could motivate relatives to learn their genetic risk (Gaff et al., 2007). We also examined participants' beliefs that they would be devastated and unable to cope if they found out negative information (i.e., anticipated negative affect; Ferrer et al., 2015), which may indicate less adaptive coping. Finally, we hypothesized that self-affirmation would mitigate these associations.

Method

Participants and procedure. Participants aged 45-65 years (n=540) recruited from the Bethesda, MD area were enrolled in a trial testing the use of genome sequencing (ClinSeq®). Data were collected from August 2012 to April 2015. The National Human Genome Research

Institute's (NHGRI) IRB approved the study. Participants completed a cross-sectional survey before receiving any genetic information, and were informed that genetic information may become available in the future although they may not learn any results. Approximately half of enrollees mentioned personal health benefits as motives for participation; the other half mentioned contributing to scientific knowledge but not personal health benefits (Facio et al., 2011; but see Sanderson et al., 2016 in which participants enrolling in a similar trial – with a primary goal of returning results—simultaneously endorsed both motivations). All provided informed consent. Additional information about the study is available elsewhere (Biesecker et al., 2009; Lewis et al., 2015). We report only measures relevant to the present analyses.

We analyzed data from 511 participants with complete data for implicit theories and sociodemographic factors used as covariates in analyses (i.e., self-reported education level, household income, gender, race [coded White vs. not White], and age). The sample size differs across analyses due to variation in missing data on other variables (range=478-511).

Measures. Implicit theories of health were measured with three items adapted from previous research examining implicit theories (Burnette, 2010; Lyons et al., 2013): "I think your {body weight/health/cancer risk} is something very basic about you that you can't change very much" (1: *Strongly disagree* to 6: *Strongly agree*). Higher scores indicated stronger fixed theories. Items were averaged (α=.694) and treated as continuous.

A list of measures is shown in Figure 1. Anticipated negative affect was assessed as the average of two items: "If I found out that my genes put me at high risk for a fatal disease, I would be devastated" and "I don't think I would be able to cope with finding out that my genes put me at high risk for a fatal disease" (1: *Strongly Disagree* to 7: *Strongly Agree*; r=.463, p<.001).

Intentions to learn and share genetic information were assessed for three types of results (Figure 1) and scored as in previous publications (Taber, Klein, Ferrer, Biesecker et al., 2015; Taber, Klein, Ferrer, Han et al., 2015). Medically actionable disease results referred to "a gene variant that predisposes you to a disease that can be prevented or treated." Nonmedically actionable disease results referred to "a gene variant that predisposes you to a disease that cannot be prevented or treated." Carrier results for a recessive condition referred to "a gene variant that does not affect your health, but that may be important to the health of other relatives, such as your children." Medically actionable disease results are more relevant to health-improvement goals and thus more likely to be associated with implicit theories.

Intentions to learn sequencing results were measured by two items: "I intend to learn such a result," 1: *Definitely no* to 5: *Definitely yes*; and "How likely is it that you will choose to learn about such a result?", 1: *Extremely unlikely* to 7: *Extremely likely*, for each of the three types of genetic results (medically actionable: r=.262, p<.001; nonmedically actionable: r=.720, p<.001; carrier status: r=.506, p<.001). Items were standardized then averaged to form independent scales for each result type. To normalize the distribution, log transformations were used. Responses were reverse-scored following transformation (see, e.g., Taber, Klein, Ferrer, Biesecker et al., 2015 for similar treatment of these variables).

Intentions to use results to change behavior were assessed by two items indicating intentions (1: *Definitely no* to 5: *Definitely yes*) and likelihood (1: *Extremely unlikely* to 5: *Extremely likely*) to use medically actionable results to "change your lifestyle/health behavior (diet, exercise, stress management)." Items were standardized and averaged. A square root transformation was applied to normalize the distribution then responses were reverse-scored.

Intentions to share sequencing results were assessed with one scale comprising the

average of six standardized items (α=.898) assessing intentions to share (1: *Definitely no* to 5: *Definitely yes*) and likelihood of sharing (1: *Extremely unlikely* to 7: *Extremely likely*) each of the three types of sequencing results with family members.

Two items assessed beliefs about gene by environment interactions (Table 1) on scales from 1: *Definitely No* to 5: *Definitely Yes*. These items were included to assess discriminant validity of implicit theories, as disagreement represents more deterministic beliefs which may be associated with fixed theories.

<INSERT TABLE 1 ABOUT HERE>

Spontaneous self-affirmation was assessed with two items: "When I feel threatened or anxious I find myself thinking about my strengths [values]," (1: *Strongly disagree* to 5: *Strongly Agree*; r=.710, p<.001). These items were taken from a longer SSA scale currently in development (Harris et al., 2017).

Results

The sample was 56.8% male and 92.6% White, with an average age of 60.8 years (*SD*=5.5). Most (78.3%) earned a household income of >$100,000, and had a college education or higher (89.4%).

Preliminary analyses. Fixed theories were endorsed by 10.8% of the sample (scores above the midpoint of the scale; *M*=2.06, *SD*=0.87). Table 1 presents means, standard deviations, and proportions of respondents endorsing implicit theory items. As expected, fixed theories of health were associated with somewhat lower endorsement of gene by environment interaction beliefs, although correlations were low (rs<-.11; Table 1). SSA was endorsed above the scale midpoint (*M*=3.52, *SD*=0.86), consistent with similar items in a nationally representative sample (Taber et al., 2016).

Effects of implicit theories. We conducted 6 linear regression analyses to test the hypothesis that greater fixed theories would be associated with less proactive and adaptive responses, controlling for sociodemographic factors (i.e., education, income, gender, race, and age). As predicted, greater fixed beliefs were associated with more negative anticipated affect (β=.19, *SE*=.07, *p*=.003, 95% confidence interval [CI] [0.065, 0.320]), lower intentions to learn results for medically actionable disease (β=-.02, *SE*=.01, *p*=.028, 95% CI [-0.040, -0.002]), lower intentions to use medically actionable disease results to change behavior (β=-.14, *SE*=.05, *p*=.004, 95% CI [-0.231, -0.043]), and lower intentions to share results (β=-.11, *SE*=.04, *p*=.008, 95% CI [-0.196, -0.029]). Implicit theories were not associated with intentions to learn nonmedically actionable or carrier status results (both *p*s > 0.10). Zero-order correlations (Table 2) showed the same pattern.

<INSERT TABLE 2 ABOUT HERE>

Moderating effects of self-affirmation. To examine whether SSA mitigates the previously-tested associations, we assessed whether implicit theories interacted with SSA to predict the four outcomes associated with implicit theories, controlling for the same set of sociodemographic factors (PROCESS macro, Hayes, 2013). Three of four interactions were significant: intentions to learn (β=.03, *SE*=.01, *p*=.002, 95% CI [0.012, 0.053]) and use (β=.16, *SE*=.05, *p*=.003, 95% CI [0.053, 0.259]) results for medically actionable disease, and intentions to share results (β=.11, *SE*=.05, *p*=.022, 95% CI [0.015, 0.197]). Simple slopes analyses indicated that, as predicted, greater fixed beliefs were associated with lower intentions for individuals *low* in SSA (1 SD<the mean; learn: β=-.05, *SE*=.01, *p*<.001, 95% CI [-0.076, -0.024]; use: β=-.28, *SE*=.07, *p*<.001, 95% CI [-0.406, -0.147]; share: β=-.21, *SE*=.06, *p*<.001, 95% CI [-0.325, -0.095]). However, there was no association for individuals *high* in SSA (1 SD above the

mean; learn: β=.01, *SE*=.01, *p*=.694, 95% CI [-0.020, 0.031]; use: β=-.01, *SE*=.07, *p*=.920, 95% CI [-0.134, 0.121]; share: β=-.03, *SE*=.06, *p*=.653, 95% CI [-0.139, 0.087]). The same pattern of effects was found when covariates were not included. The pattern of results for intentions to learn results for medically actionable disease is shown in Figure 2.

<INSERT FIGURE 2 ABOUT HERE>

Study 1 Discussion

Individuals with entity beliefs about health perceived less utility of genetic information for medically actionable disease. As predicted, this did not happen for participants high in SSA, supporting the idea that such implicit theories are associated with defensiveness. Of note, these three interactions held even when controlling for beliefs about gene by environment interactions in addition to the main set of covariates, which suggests that implicit theories are not simply a proxy for these beliefs. However, when we controlled for these beliefs in the 6 initial regressions, the association of implicit theories with intentions to learn medically actionable results became nonsignificant (*p*=.060), whereas the item "Even if people have a variant in a gene that affects their risk of a disease, they may not develop that disease" was a significant predictor (β=.029, *p*=.009).

Study 2

Study 1 showed that stronger fixed theories about health are associated with lower (though still high) intentions to engage with risk information for medically actionable disease, although not among people higher in SSA. If fixed theories are associated with less interest in learning risk information, how might such theories influence reactions to learning that genetics cause health problems? In Study 2, overweight women received information about genetics or behavior as risk factors for weight, and we determined how fixed versus growth theorists reacted

to such information. We also assessed whether SSA facilitated more adaptive use of the information. We expected that among those higher in SSA, receiving information about risk factors for overweight (regardless of information type) would be associated with lower endorsement of fixed theories. Behavioral information might be threatening because it implicates personal behavior in weight gain, and genetic information could promote beliefs that weight cannot change; SSA should offset these effects.

We report two primary sets of analyses (see Figure 1). First, we examined whether participants' baseline implicit theories were differentially associated with self-efficacy, motivation, and intentions to diet and exercise depending on whether they received genetic versus behavioral explanations for weight. Second, we tested whether people showed lower increases in fixed beliefs about weight following provision of information about risk factors if they were higher in SSA.

Method

Participants and procedure. Data were taken from an experiment conducted from July 2013 to April 2014 with a 2 (information about disease risk factors: genetic, behavior) x 2 (induced emotion: fear, anger) design. Emotion type was not included in analyses because it was irrelevant to our hypotheses and controlling for the main effect of emotion type did not change the pattern of results. Overweight and obese women (≥25 BMI; 18 to 50 years old; n=201) participated in this study, in which a virtual reality (VR) physician provided information that genetics or behavior were primary risk factors for overweight (i.e., causal information). Participants wore a head-mounted VR display that delivered 3D, stereoscopic stimuli, through which they verbally interacted with the physician during a simulated clinical visit. A VR simulation allowed for ecological validity and psychological realism while retaining strict

experimental control (Blascovich et al., 2002; Persky, 2011). In both conditions, the physician discussed the importance of weight management and the link between overweight and breast cancer risk. Participants in the genetic condition were told that genetics and DNA confer risk for increased body weight, and that this may occur through multiple means including metabolism and influences on behavior (e.g., which foods people like to eat, exercise enjoyment). Participants in the behavioral condition were told that their behavior and the environment, including what they eat and how much they exercise, confer risk for increased body weight and that, when people lose weight, their bodies try to return to a stable point (this information was added so weight loss would not seem inherently more difficult in either condition). After providing information about the causes of overweight, the physician emphasized the importance of physical activity and nutrition in both conditions. Additional methodological details are provided elsewhere (Beekman, Ferrer, Klein, & Persky, 2015; Persky, Ferrer, & Klein, 2016), and the doctor's script is included in Supplemental Materials.

All participants provided informed consent and NHGRI's IRB approved the study. We report only measures relevant to the present analyses. Participants were surveyed before (Baseline) and after (Post) the manipulation. Analyses were restricted to 197 participants who completed all items.

Measures. At Baseline and Post, implicit theories about body weight were measured with: "Body weight is something basic about a person that they can't change very much" (HINTS 4 Cycle 2; 1: *Strongly agree* to 4: *Strongly disagree* at Baseline, reverse-scored; 1: *Strongly disagree* to 7: *Strongly agree* at Post); higher scores indicate stronger fixed beliefs. [The response scales for implicit theories differed between Baseline and Post because of a programming error.] At Baseline and Post, self-efficacy (average of three items: "I feel as if I

could take the right actions to achieve a healthy weight/eat a healthier diet/get more physical activity"; 1: *Strongly disagree* to 7: *Strongly agree*; α_{Time1}=.866, α_{Time2}=.811) and motivation "to achieve a healthy weight" (1: *Not at all motivated* to 7: *Extremely motivated*) were assessed. At Post only, intentions to change diet were assessed as the average of two items: "I intend to make changes to my diet in the next 6 months" (1: *Strongly disagree* to 7: *Strongly agree*) and "How likely is it you will try to change your diet in the next 6 months? (1: *Very unlikely* to 7: *Very likely*; r=.628, p<.001). Intentions to increase exercise were assessed with two items in which "increase exercise" and "get more exercise" replaced "make changes to my diet" and "change your diet," respectively (r=.622, p<.001). At Baseline only, SSA was assessed as the average of three items: "When I find myself threatened or anxious by people or events, I find myself thinking about [my strengths/my principles/what I stand for]" (1: *Disagree completely* to 7: *Agree completely*; α=.882). At Baseline only, genetic causal attributions were assessed with, "To what extent do you agree or disagree that each of the following factors cause or contribute to your body weight? Genetics" (1: *Strongly disagree* to 7: *Strongly agree*). Behavioral causal attributions were assessed as the average of, "Eating too much or too many unhealthy foods," "not exercising enough," and "sitting and not moving enough" (α=.748). All items were treated as continuous.

Square root transformations were used to normalize the distribution of implicit theories at Post, self-efficacy at Baseline, self-efficacy at Post, and intentions to exercise.

Self-reported height and weight were used to calculate BMI (CDCP, 2015), which was coded 1=obese, 0=overweight to reduce the influence of outliers. Participants also reported race (1=African American, 0=not African American), education (1=college education or higher, 0=less than college education), and age.

Results and Discussion

Participants were on average 35.2 years old (*SD*=9.2; range=20-50). Most were African American (50.3%). Over half were obese (56.9%) and had a college degree or higher (64.5%). At Baseline, fixed beliefs about weight (*M*=1.70 of 4; *SD*=.76) were endorsed by 15.2%, with lower endorsement at Post (*M*=2.30 of 7; *SD*=1.48; 10.6% agreed; $\chi^2(1)=19.10$, $p<.001$), consistent with the physician emphasizing the health benefits of physical activity and nutrition in both conditions. Self-efficacy, motivation, and intentions to diet and exercise were all highly endorsed (Self-efficacy before transformation: Baseline: *M*=5.67, *SD*=1.09; Post: *M*=6.12, *SD*=0.89; Motivation: Baseline: *M*=5.23, *SD*=1.22; Post: *M*=6.00, *SD*=0.90; Intentions to diet before transformation: *M*=6.00, *SD*=1.10; Intentions to exercise: *M*=6.16, *SD*=1.01). Baseline implicit theories were associated with greater endorsement of genetic but not behavioral causal attributions (Table 1).

We conducted cross-sectional partial correlations to determine the extent to which implicit theories of weight were associated with outcomes, controlling for BMI, education, race, and age. At Baseline, greater fixed beliefs were associated with lower self-efficacy ($r=-.285$, $p<.001$) and lower motivation ($r=-.182$, $p=.011$). At Post, greater fixed beliefs were associated with lower self-efficacy ($r=-.192$, $p=.008$), motivation ($r=-.242$, $p=.001$), and intentions to diet ($r=-.197$, $p=.006$) and exercise ($r=-.206$, $p=.004$)—consistent with Study 1.

Implicit theories as a predictor of self-efficacy, motivation, and intentions. We next used hierarchical linear regressions to test whether Baseline implicit theories moderated the effect of information type (behavioral=0, genetic=1) on self-efficacy, motivation, and intentions at Post (four analyses total). We controlled for race (AA=1, not AA=0) because more African Americans were randomly assigned to the behavioral (57.0%) than the genetic condition (43.3%;

$\chi^2(1)=3.697$, $p=.055$), although implicit theories at Baseline did not differ by race ($t(195)= 0.719$, $p=.473$), and the pattern of results remained the same when race was not covaried. Education, age, and BMI were not covaried because they were not significantly associated with Baseline implicit theories nor did they differ by condition.

Regression results are shown in Table 3. Stronger fixed theories at Baseline were significantly associated with lower motivation and lower intentions to diet and exercise, but not with self-efficacy. Of greater interest, the implicit theory by causal information type interaction was significant for two of four outcomes: motivation and diet intentions. Specifically, among participants given behavioral explanations for weight, greater fixed beliefs were associated with lower motivation (β=-0.32, SE=.12, p=.009,) and lower intentions to diet at Post (β=-.43, SE=.14, p=.003). However, among participants given genetic explanations, implicit theories were not associated with motivation (β=0.05, SE=.12, p=.681) or diet intentions (β=.01, SE=.14, p=.970). Race was a significant predictor of self-efficacy and diet/exercise intentions, but not motivation. As shown in Figure 3, participants with lower fixed beliefs who were given behavioral explanations for overweight reported greater motivation to achieve a healthy weight and intentions to change diet.

<INSERT TABLE 3 ABOUT HERE>

<INSERT FIGURE 3 ABOUT HERE>

In four regressions in which baseline SSA was included as a moderator, there were no significant 3-way interactions of SSA, information type, and implicit theories predicting self-efficacy, motivation, or intentions to diet or exercise. Thus, the effects of implicit theories and information type were not modified by SSA.

SSA as a predictor of adopting implicit theories. We used hierarchical linear

regressions to test whether SSA moderated the effect of information type (i.e., behavioral or genetic) on implicit theories at Post. Implicit theories at Baseline were covaried, as was race because it differed by information condition (as reported previously), and because African Americans were higher in SSA (M=4.73, SD=1.63) than other respondents (M=4.10, SD=1.50; t(195)= -2.84, p=.005); the pattern of effects did not differ when race was not covaried. Education, age, and BMI were not covaried because they were not significantly associated with SSA nor did they differ by condition.

Contrary to the hypothesis that participants given information about risk factors for weight would have less fixed beliefs if they were higher versus lower in SSA, the main effect of SSA was nonsignificant (β=0.01, SE=.02, p=.674). However, a significant causal information type by SSA interaction (β=-0.08, SE=.04, p=.026) indicated that among participants given genetic explanations for weight, greater SSA was associated with weaker fixed theories at Post (β=-0.07, SE=.03, p=.009). Among participants given behavioral weight explanations, SSA was not associated with implicit theories (β=.01, SE=.02, p=.674). Thus, SSA seems to reduce the likelihood that information about genetics as a risk factor for obesity will lead to fixed beliefs. There were significant main effects of Baseline implicit theories (β=0.26, SE=.04, p<.001) and Race (β=-0.14, SE=.06, p=.017). The main effect of information type was nonsignificant (β=-0.05, SE=.06, p=.424), perhaps because the importance of lifestyle changes was emphasized in both conditions.

General Discussion

Across two studies, we investigated how self-affirmation and implicit theories about body weight and health were linked to engagement with genetic risk information. We found initial evidence that fixed theories were associated with reduced (though still high) interest in learning

risk information for medically actionable disease and subsequently lower intentions to engage in behavior to mitigate this risk. These findings contribute to mounting evidence that holding fixed beliefs may be maladaptive (Paunesku et al., 2015; Burnette & Finkel, 2012).

One goal of providing people with health risk information is to motivate risk-mitigating behavior and ultimately improve health. This goal is less likely to be achieved if (1) people decline to learn information about their risk, and (2) those who learn risk information subsequently adopt beliefs that they cannot change relevant risk factors. Further, people who believe they cannot change their risk factors may be reluctant to change their behavior after learning risk information. Across two studies, our data suggest that holding stronger health-related fixed theories (i.e., beliefs that health or body weight cannot change) may decrease the likelihood of learning and benefiting from health risk information. Importantly, the association of implicit theories and maladaptive responding was weakened for people who naturally self-affirm, suggesting potentially beneficial effects of self-affirmation.

In Study 1, people holding fixed theories expressed less interest in learning, using, and sharing genetic risk information, but only for diseases for which preventable actions could be taken (i.e., medically-actionable). These findings are consistent with data from studies showing that fixed theories promote less active coping (Burnette, 2010; Burnette et al., 2013), and active coping attempts are more relevant to diseases for which preventive action can be taken.

One barrier to realizing the full utility of providing risk information is that people who believe they cannot change risk factors may be less motivated to subsequently engage in risk-mitigating behaviors. We examined this barrier in an experiment (Study 2). We found that motivation and intentions to diet differed according to fixed beliefs for women given behavioral information but not for women given genetic information. More specifically, overweight women

with lower fixed beliefs who received information that behavior caused overweight reported higher motivation to achieve a healthy weight and higher intentions to diet compared to women given genetic attributions and women with less fixed beliefs given behavioral information. For women with relatively *higher* fixed beliefs, the pattern of results is consistent with evidence that receiving obesity genetic feedback does not decrease weight control intentions (Meisel et al., 2015). For women with relatively *lower* fixed beliefs, the pattern of results is consistent with some studies that have shown genetic information about weight to be demotivating (Dar-Nimrod, Cheung, Ruby, & Heine, 2014; Persky, Ferrer, & Klein, 2015). Thus, how women respond to behavioral risk information about overweight may differ according to their implicit theories about the malleability of overweight.

Here, for women with higher baseline beliefs that their weight cannot change, being told that their weight was largely due to behavioral factors may have been interpreted as threatening feedback that they were responsible for past weight loss failures. Alternatively, messages about behavioral factors may be dismissed because they do not include novel information. Thus, as the current study showed, messages emphasizing behavioral factors may be less likely to promote change for women higher in fixed beliefs than for women lower in fixed beliefs, consistent with the argument that having fixed beliefs promotes defensiveness to self-threatening information. Importantly, this effect was found for only two of four outcomes examined (i.e., motivations to maintain a healthy weight and intentions to diet), suggesting a need for replication. Further, describing genetics as a risk factor for overweight in clinical discussions about weight management may be equally effective for women regardless of their implicit theories about overweight.

Another barrier to realizing the full utility of providing risk information is that people

who learn they are at high risk could become fatalistic, which could manifest as fixed theories. Our experimental data showed that women told of a genetic basis for being overweight were less likely to adopt fixed theories of weight when they were higher in SSA. Self-affirmation reduces defensiveness, and we found an interactive effect of SSA and implicit theories: fixed theories were associated with lower intentions to learn genetic information only among individuals lower in SSA. This finding supports the argument that fixed theories may be associated with defensiveness. In addition, among people with fixed beliefs, SSA was associated with increased interest in learning and using risk information. Indeed, SSA can mitigate the association of information avoidance (Taber, Klein, Ferrer, Lewis, et al. 2015), perceived ambiguity (Taber, Klein, Ferrer, Han, et al. 2015), and anticipated negative affect (Ferrer et al., 2015) with lower intentions to learn risk information.

These findings suggest multiple routes to promoting behavioral change among people who hold fixed theories. One is to promote self-affirmation—a malleable construct—among people with fixed theories. Another is to promote growth theories, as implicit theories are also malleable (Burnette & Finkel, 2012). However, it might be beneficial to test whether growth theories have any negative consequences: if people believe that weight can change (or is easy to change), they might be more likely to attribute others' overweight to personal behavior, potentially leading to stigmatization and discrimination.

Neither study included a battery of control-related constructs, such as personal control over prevention, perceived behavioral control, or causal attributions. However, implicit theories about body weight seem to be distinct from dispositional self-control, dieting self-confidence, general health locus of control, and self-efficacy (Burnette, 2010; Fitz, Kaufman, & Moore, 2015). Implicit theories are also conceptually similar to but distinct from causal attributions. This

is evidenced in part by our finding that causal explanations for weight interacted with implicit theories to influence diet intentions in Study 2. Inconsistent evidence exists as to whether and how causal beliefs about obesity are associated with health behavior performance and cognitions (Nguyen, Oh, Moser, & Patrick., 2014), perhaps because causal attributions do not assess perceived control. Implicit theories, which suggest whether one's efforts will change that characteristic, may be more predictive of intentions.

Limitations

In Study 1, implicit theories assessed whether participants believed "your" body—rather than "a person's"— weight can change. However, endorsement of implicit theories was similar, and findings consistent, across studies despite minor wording differences. Although fixed beliefs about health and body weight were infrequently endorsed, they were associated with several important outcomes. Additionally, one-item measures of implicit theories were used rather than the full scale (see Burnette 2010). Single items were used because they were part of larger studies with other goals and multiple other measures. In prior research, the full 6-item scale showed high reliability (α=.82; Burnette, 2010) and all items loaded on one factor (α=.83; Lyons et al., 2013).

Participants in Study 1 were high in socioeconomic status, consistent with other samples in genetic testing studies (Hensley et al., 2011), but not generalizable to the general public. We assessed intentions but not behavior, which is common when behavior may be expensive (e.g., receipt of genomic sequencing) or difficult to assess (e.g., long-term weight loss). Further, in Study 1 intentions were skewed towards a desire to learn results and to use results to change behavior; this is unsurprising as many participants reported participating to learn personal health information. Given restricted range, we expect that effects might be stronger in a sample with

greater variability in intentions. However, we should exercise caution in extrapolating the current findings to a samples with less interest in learning genetic risk information.

Conclusion

Research has shown that implicit theories have powerful consequences in numerous domains. We expanded the study of implicit theories to the domain of genetic risk information. Our results suggest that growth (versus fixed) theories are beneficial in motivating risk-mitigating behavioral intentions and interest in learning personalized risk information (albeit in a sample with high intentions to learn genetic risk information in Study 1). Using differing samples and methodology, our data suggest that implicit theories and self-affirmation may have interactive effects on how people engage with and respond to risk information. Self-affirmation is malleable and holds promise as a way to decrease the negative consequences of fixed beliefs on motivation and behavior.

References

Arciszewski, T., Berjot, S., & Finez, L., 2012. Threat of the thin-ideal body image and body malleability beliefs: effects on body image self-discrepancies and behavioral intentions. Body Image, 9, 334-341.

Aspinwall, L.G., Stump, T.K., Taber, J.M., Kohlmann, W., Leaf, S.L., & Leachman, S.A., 2015. Impact of melanoma genetic test reporting on perceived control over melanoma prevention. J. Behav. Med., 38, 754-65.

Aspinwall, L.G., Taber, J.M., Kohlmann, W., & Leachman, S.A., 2013. Genetic counseling and cancer, in: Carr, B.I., Steel, J. (Eds.), Psychological Aspects of Cancer: A Guide to Emotional and Psychological Consequences of Cancer, Their Causes and Their Management. Springer, pp. 61-64.

Aspinwall, L.G., & Taylor, S.E., 1997. A stitch in time: self-regulation and proactive coping. Psychol. Bull., 121, 417-436.

Beekman, J.B., Ferrer, R.A., Klein, W.M.P., & Persky, S., 2016. Effects of perceived weight discrimination on willingness to adopt unhealthy behaviours: influence of genomic information. Psychol. Health., 31: 334-348.

Biesecker, B.B., Klein, W.M.P., Lewis, K.L., Fisher, T.C., Wright, M.F., Biesecker, L.G., & Han, P.K., 2014. How do research participants perceive "uncertainty" in genome sequencing? Genet. Med., 16, 977-980.

Biesecker, L.G., Mullikin, J.C., Facio, F.M., Turner, C., Cherukuri, P.F., Blakesley, R.W.,...Green, E.D., 2009. The ClinSeq Project: piloting large-scale genome sequencing for research in genomic medicine. Genome Res., 19, 1665-1674.

Blascovich, J., Loomis, J., Beall, A., Swinth, K., Hoyt, C., & Bailenson, J., 2002. Immersive virtual environment technology as a research tool for social psychology. Psychol. Inquiry, 13, 103-125.

Burnette, J.L., 2010. Implicit theories of body weight: Entity beliefs can weigh you down. Pers.

Soc. Psychol. Bull., 36, 410-422.

Burnette, J.L., & Finkel, E J., 2012. Buffering against weight gain following dieting setbacks: An implicit theory intervention. J. Exp. Soc. Psychol., 48, 721-725.

Burnette, J.L., O'Boyle, E.H., VanEpps, E.M., Pollack, J.M., & Finkel, E.J., 2013. Mind-sets matter: A meta-analytic review of implicit theories and self-regulation. Psychol. Bull., 139, 655-701.

Chao, S., Roberts, J.S., Marteau, T.M., Silliman, R., Cupples, L.A., & Green, R.C., 2008. Health behavior changes after genetic risk assessment for Alzheimer disease: The REVEAL study. Alzheimer Dis. Assoc. Disord., 22, 94–97.

Collins, F.S., & Varmus, H., 2015. A new initiative on precision medicine. N. Engl. J. Med., 372, 793-795.

Condit, C.M., & Shen, L., 2011. Public understanding of risks from gene-environment interaction in common diseases: implications for public communications. Public Health Genomics, 14, 115-124.

Conradt, M., Dierk, J.-M., Schlumberger, P., Albohn, C., Rauh, E., Hinney, A.,...Rief, W., 2009. A consultation with genetic information about obesity decreases self-blame about eating and leads to realistic weight loss goals in obese individuals. J. Psychosom. Res., 66, 287-295.

Cornil, Y., & Chandon, P., 2013. From fan to fat? Vicarious losing increases unhealthy eating, but self-affirmation is an effective remedy. Psychol. Sci., 24, 1936-1946.

Dar-Nimrod, I., Cheung, B.Y., Ruby, M.B., & Heine, S.J., 2014. Can merely learning about obesity genes affect eating behavior? Appetite, 81, 269-76.

Dweck, C., 2006. Mindset: The new psychology of success: Random House.

Dweck, C.S., & Leggett, E.L., 1988. A social-cognitive approach to motivation and personality. Psychol. Rev., 95, 256-273.

Epton, T., Harris, P.R., Kane, R., van Koningsbruggen, G.M., & Sheeran, P., 2014. The impact of self-affirmation on health-behavior change: A meta-analysis. Health Psychol., 34, 187-

196.

Facio, F.M., Brooks, S., Loewenstein, J., Green, S., Biesecker, L.G., & Biesecker, B.B., 2011. Motivators for participation in a whole-genome sequencing study: Implications for translational genomics research. European Journal of Human Genetics, 19, 1213–1217.

Ferrer, R.A., Taber, J.M., Klein, W.M.P., Harris, P., Lewis, K., & Biesecker, L.G., 2015. The role of current affect, anticipated affect, and spontaneous self-affirmation in decisions to receive self-threatening genetic risk information. Cog. Emot., 29, 1456-1465.

Fitz, C.C., Kaufman, A., Moore, P.J., 2015. Lay theories of smoking and young adult nonsmokers' and smokers' smoking expectations. J. Health Psychol., 20, 438-445.

Gaff, C.L., Clarke, A.J., Atkinson, P., Sivell, S., Elwyn, G., Iredale, R.,...& Edwards, A., 2007. Process and outcome in communication of genetic information within families: a systematic review. Eur. J. Hum. Genet., 15, 999-1011.

Harris, P.R., Griffin, D.W., Napper, L., Schuez, B., Stride, C., & Bond, R., 2017. Individual differences in self-affirmation: Distinguishing self-affirmation from positive self-regard. In preparation.

Hayes, A.F., 2013. Introduction to mediation, moderation, and conditional process analysis: A regression-based approach. New York, NY US: Guilford Press.

Health Information National Trends Survey 4 Cycle 2. https://hints.cancer.gov/question-details.aspx?PK_Cycle=5&qid=1250 (accessed 17.07.27)

Hensley, Alford S., McBride, C.M., Reid, R.J., Larson, E.B., Baxevanis, A.D., & Brody, L.C., 2011. Participation in genetic testing research varies by social group. Public Health Genomics, 14, 85–93.

Khoury, M.J., Gwinn, M., Yoon, P.W., Dowling, N., Moore, C.A., & Bradley, L., 2007. The continuum of translation research in genomic medicine: How can we accelerate the appropriate integration of human genome discoveries into health care and disease prevention? Genet. Med., 9, 665-674.

Lewis, K.L., Han, P.K., Hooker, G.W., Klein, W.M., Biesecker, L.G., & Biesecker, B.B., 2015. Characterizing Participants in the ClinSeq Genome Sequencing Cohort as Early Adopters of a New Health Technology. PLoS One, 10, e0132690.

Lyons, C., Kaufman, A.R., & Rima, B., 2013. Implicit theories of the body among college women: Implications for physical activity. J. Health Psychol., 20, 1142-1153.

Meisel, S.F., Beeken, R.J., van Jaarsveld, C.H., & Wardle, J., 2015. Genetic susceptibility testing and readiness to control weight: Results from a randomized controlled trial. Obesity, 23, 305-312.

Molden, D.C., & Dweck, C.S., 2006. Finding "meaning" in psychology: A lay theories approach to self-regulation, social perception, and social development. Am. Psychol., 61, 192-203.

Nguyen, A.B., Oh, A., Moser, R.P., & Patrick, H., 2014. Perceptions of the roles of behaviour and genetics in disease risk: Are they associated with behaviour change attempts. Psychol. Health, 30, 336-353.

Parrott, R., Silk, K., Weiner, J., Condit, C., Harris, T., & Bernhardt, J., 2004. Deriving lay models of uncertainty about genes' role in illness causation to guide communication about human genetics. J. Comm., 54, 105–122.

Paunesku, D., Walton, G.M., Romero, C., Smith, E.N., Yeager, D.S., & Dweck, C.S., 2015. Mind-set interventions are a scalable treatment for academic underachievement. Psychol. Sci. 26, 784-793.

Persky, S., 2011. Employing immersive virtual environments for innovative experiments in health care communication. Patient Educ. Couns., 82, 313-317.

Persky, S., Ferrer, R.A., & Klein, W.M.P., 2016. Genomic information may inhibit weight-related behavior change inclinations among individuals in a fear state. Ann. Behav. Med., 50, 452-459.

Persky, S., Ferrer, R.A., & Klein, W.M.P., 2016. Nonverbal and paraverbal behavior in (simulated) medical visits related to genomics and weight: a role for emotion and race. J.

Behav. Med., 39, 804-814.

Persky, S., & Street Jr, R.L., 2015. Evaluating approaches for communication about genomic influences on body weight. Ann. Behav. Med., 49, 675-684.

Rief, W., Conradt, M., Dierk, J.-M., Rauh, E., Schlumberger, P., Hinney, A., & Hebebrand, J., 2007. Is information on genetic determinants of obesity helpful or harmful for obese people? A randomized clinical trial. J. Gen. Int. Med., 22, 1553-1559.

Sanderson, S.C., Linderman, M.D., Suckiel, S.A., Diaz, G.A., Zinberg, R.E., Ferryman, K.,...& Schadt, E.E., 2016. Motivations, concerns and preferences of personal genome sequencing research participants: Baseline findings from the HealthSeq project. Eur. J. Hum. Genet., 24, 14-20.

Sanderson, S., Persky, S., & Michie, S., 2010. Psychological and behavioral responses to genetic test results indicating increased risk of obesity: Does the causal pathway from gene to obesity matter? Public Health Genomics, 13, 34-47.

Senior, V., & Marteau, T.M., 2007. Causal attributions for raised cholesterol and perceptions of effective risk-reduction: Self-regulation strategies for an increased risk of coronary heart disease. Psychol. Health, 22, 699-717.

Shen, L., Condit, C.M., & Wright, L., 2009. The psychometric property and validation of a fatalism scale. Psychol. Health, 24, 597-613.

Steele, C.M., 1988. The psychology of self-affirmation: Sustaining the integrity of the self. Adv.Exp. Soc. Psychol., 21, 261-302.

Sweeny, K., Ghane, A., Legg, A.M., Huynh, H.P., & Andrews, S.E., 2014. Predictors of genetic testing decisions: A systematic review and critique of the literature. J. Genet. Couns., 23, 263-288.

Taber, J.M., Howell, J.L., Emanuel, A.S., Klein, W.M.P., Ferrer, R.A., & Harris, P.R., 2016. Associations of spontaneous self-affirmation with health care experiences and health information seeking in a national survey of US adults. Psychol. Health, 31, 292-309.

Taber, J.M., Klein, W.M.P., Ferrer, R.A., Biesecker, B.B., Lewis, K.L., & Biesecker, L.G., 2015. Dispositional optimism and perceived risk interact to predict intentions to learn genome sequencing results. Health Psychol., 34, 718-728.

Taber, J.M., Klein, W.M.P., Ferrer, R.A., Han, P., Lewis, K.L., Biesecker, L.G., & Biesecker, B.B., 2015. Perceived ambiguity as a barrier to intentions to learn genome sequencing results. J. Behav. Med., 38, 715-726.

Taber, J.M., Klein, W.M.P., Ferrer, R.A., Lewis, K.L., Harris, P.R., Shepperd, J.A., & Biesecker, L.G., 2015. Information avoidance tendencies, threat management resources, and interest in genetic sequencing feedback. Ann. Behav. Med., 49, 616-621.

Williams, J.K., Erwin, C., Juhl, A., Mills, J., Brossman, B., & Paulsen, J.S., 2010. Personal factors associated with reported benefits of Huntington disease family history or genetic testing. Genet. Test. Mol. Biomarkers, 14, 629–636.

Table 1. Endorsement of implicit theories and correlations with gene by environment interaction beliefs

			Correlation with implicit health-related theory (I think your body weight [cancer risk/health] is something very basic about you that you can't change very much)	
Study 1 (*n*=511)	M (*SD*)	% endorsing	*r*	*p*
Implicit theory of body weight	1.91 (1.10)	11.3	--	--
Implicit theory of cancer risk	2.50 (1.25)	21.9	--	--
Implicit theory of health	1.77 (0.93)	5.5	--	--
Implicit health-related theory (average)	2.06 (0.87)	10.8	--	--
Even if people have a variant in a gene that affects their risk of a disease, they may not develop that disease.	4.25 (0.78)	87.5	-0.104	.020
People's health habits, like diet and exercise, can affect whether or not their genes cause diseases.	3.80 (1.09)	70.2	-0.077	.084
			Correlation with implicit theory at Baseline (Body weight is something basic about a person that they can't change very much)	
Study 2 (*n*=197)	M (*SD*)	% endorsing	*r*	*P*
Implicit theory of body weight, Baseline	1.69 (0.76)	15.2		
To what extent do you agree/disagree that genetic factors contribute to your body weight?	4.72 (1.64)	58.9	0.212	.003
To what extent do you agree or disagree that eating too much or too many unhealthy foods [lack of exercise/sitting/ not moving enough] contributes to your body weight?	5.73 (1.24)	87.3	-0.037	.609

Note. For Study 1 and Study 2, endorsement is assessed as all responses agreeing with an item or above the scale midpoint. For all implicit theory items, endorsement is the proportion endorsing fixed theories.

Table 2. Correlations among Study 1 items, *n*s range from 478 to 508.

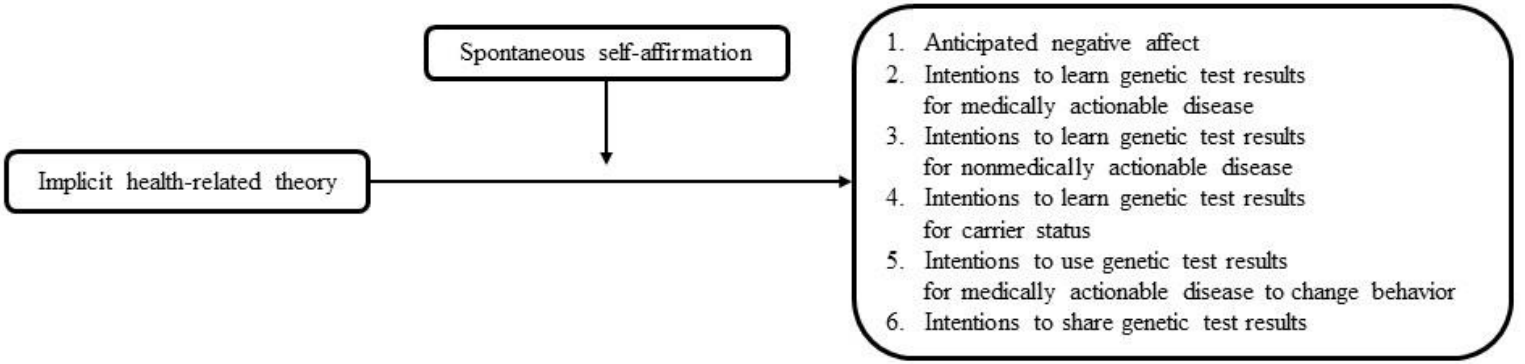
	1	2	3	4	5	6	7	8	9	10
1. Implicit health-related theory	1	-.03	-.10*	-.08	.13**	-.11*	-.05	-.04	-.14**	-.12*
2. Spontaneous self-affirmation		1	.04	.07	-.20**	.09*	.08	.11*	.14**	.13**
3. Even if people have a variant in a gene that affects their risk of a disease, they may not develop that disease.			1	.21**	-.12**	.14**	.04	.07	-.002	.07
4. People's health habits, like diet and exercise, can affect whether or not their genes cause diseases.				1	-.03	.03	.02	.04	.02	.002
5. Anticipated negative affect					1	-.19**	-.32**	-.31**	-.04	-.26**
6. Intentions to learn medically actionable disease results						1	.56**	.62**	.16**	.21**
7. Intentions to learn nonmedically actionable disease results							1	.84**	.08	.32**
8. Intentions to learn carrier results								1	.11*	.36**
9. Intentions to use medically actionable disease results to change lifestyle/health behaviors									1	.38**
10. Intentions to share results										1

**$p<.01$, *$p<.05$

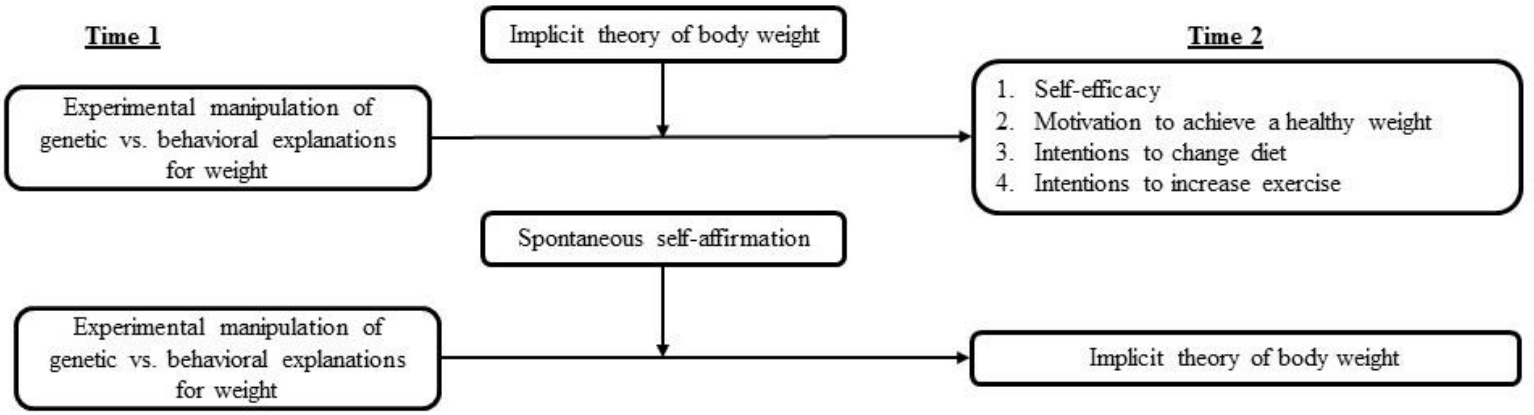
Table 3. Statistics from four hierarchical linear regressions from Study 2 testing whether implicit theories influence responses to causal information about weight.

	Self-efficacy			Motivation to achieve healthy weight			Intentions to diet			Intentions to exercise		
Predictor	*b*	*SE*	*p*	*b*	*SE*	*p*	*b*	*SE*	*p*	*B*	*SE*	*p*
Race	-0.14	.04	.001	0.19	.13	.145	0.53	.15	.001	-0.11	.05	.026
Implicit theories, Baseline	0.07	.04	.112	-0.32	.12	.009	-0.43	.14	.003	0.11	.05	.022
Causal information	-0.07	.10	.499	-0.57	.31	.066	-0.65	.37	.080	0.08	.12	.486
Causal information × Implicit theories, Baseline	0.02	.06	.675	0.37	.17	.029	0.44	.20	.028	-0.08	.06	.224

Note. Causal information was coded as behavioral information=0, genetic information=1.



Study 1: Cross-sectional survey data from a genome sequencing trial were used to examine whether implicit theories about health were associated with consequential outcomes of intentions to learn and use genetic risk information, and whether spontaneous self-affirmation moderated these associations. Cross-sectional data do not allow us to test causality, and thus direction of the arrows suggests hypothesized associations that cannot be tested with the present data.



Study 2: Participants were given surveys before and after an experimental manipulation in which they were informed of genetic or behavioral explanations for weight by a virtual reality doctor.

Figure 1. Overview and design of studies

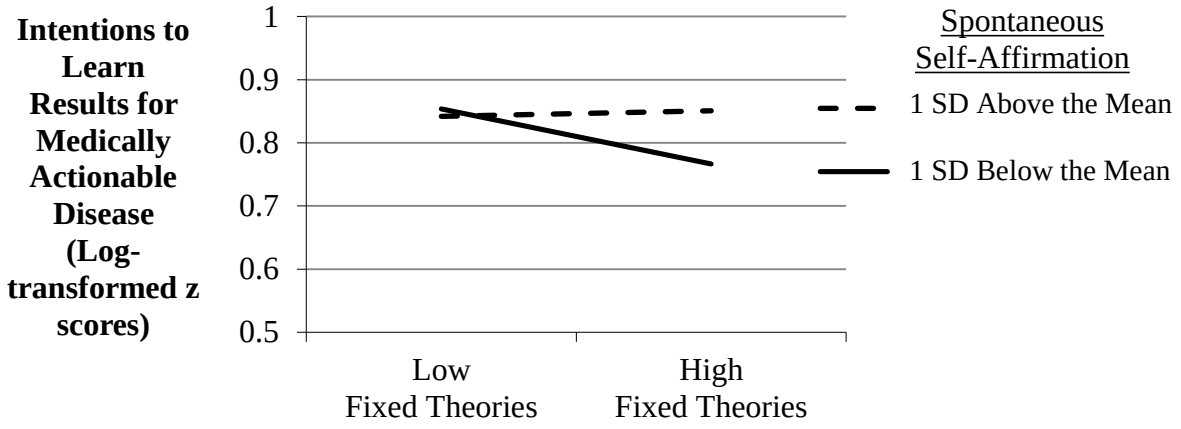


Figure 2. Interactive effects of implicit theories and self-affirmation on intentions to learn results for medically actionable disease (Study 1).

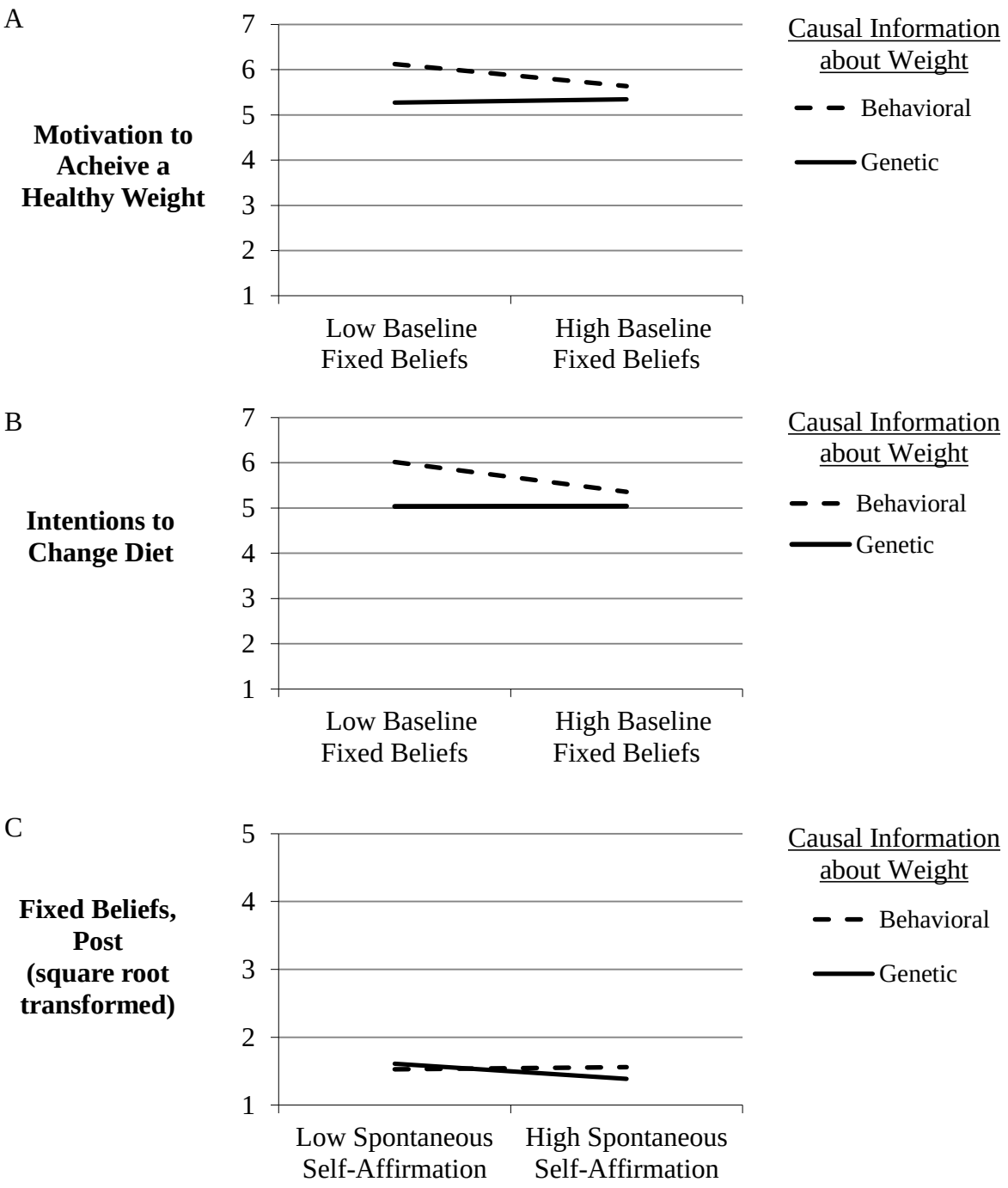


Figure 3. Interactive effects of behavioral or genetic causal information about weight and baseline implicit theories on motivation to achieve a healthy weight (3a) and intentions to change diet (3b) and of behavioral or genetic causal information about weight and spontaneous self-affirmation on implicit theories at the Post assessment (3c) (Study 2).